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# Health Effects Assessment for 1,1,2-Trichloroethane

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# HEALTH EFFECTS ASSESSMENT FOR 1,1,2-TRICHLOROETHANE

U.S. Environmental Protection Agency
Office of Research and Development
Office of Health and Environmental Assessment
Environmental Criteria and Assessment Office
Cincinnati, OH 45268

U.S. Environmental Protection Agency
Office of Emergency and Remedial Response
Office of Solid Waste and Emergency Response
Washington, DC 20460

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16. ABSTRACT

This document represents a brief, quantitatively oriented scientific summary of health effects data. It was developed by the Environmental Criteria and Assessment Office to assist the Office of Emergency and Remedial Response in establishing chemical-specific health-related goals of remedial actions. If applicable, chemical-specific subchronic and chronic toxicity interim acceptable intakes are determined for systemic toxicants, or $q_1^*$ values are determined for carcinogens for both oral and inhalation routes.

A $q_1^*$ was determined for 1,1,2-trichloroethane based on oral exposure.

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## DISCLAIMER

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## PREFACE

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with 1,1,2-trichloroethane. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. Pertinent toxicologic and environmental data were located through on-line literature searches of the Chemical Abstracts, TOXLINE, CANCERLINE and the CHEMFATE/DATALOG data bases. The basic literature searched supporting this document is current up to September, 1984. Secondary sources of information have also been relied upon in the preparation of this report and represent large-scale health assessment efforts that entail extensive peer and Agency review. The following Office of Health and Environmental Assessment (OHEA) sources have been extensively utilized:

U.S. EPA. 1980b. Ambient Water Quality Criteria for Chlorinated Ethanes. Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 440/5-80-029. NTIS PB 81-117400. (Cited in U.S. EPA, 1982)

U.S. EPA. 1982. Hazard Profile on 1,1,2-trichloroethane. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA for the Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1983b. Review of Toxicologic Data in Support of Evaluation for Carcinogenic Potential of: 1,1,2-Trichloroethane. Prepared by the Carcinogen Assessment Group, OHEA, Washington, DC for the Office of Solid Waste and Emergency Response, Washington, DC.

The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data were available. Values were not derived or larger uncertainty factors were employed when the variable data were limited in scope tending to generate conservative (i.e., protective) estimates. Nevertheless, the interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed.

Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, the AIS or acceptable intake subchronic, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval (i.e., for an interval that does not constitute a significant portion of the lifespan). This type of exposure estimate has not been extensively used or rigorously defined, as previous risk assessment efforts have been primarily directed towards exposures from toxicants in ambient air or water where lifetime exposure is assumed. Animal data used for AIS estimates generally include exposures with durations of 30-90 days. Subchronic human data are rarely available. Reported exposures are usually from chronic occupational exposure situations or from reports of acute accidental exposure.

The AIC, acceptable intake chronic, is similar in concept to the ADI (acceptable daily intake). It is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan [see U.S. EPA (1980a) for a discussion of this concept]. The AIC is route specific and estimates acceptable exposure for a given route with the implicit assumption that exposure by other routes is insignificant.

Composite scores (CSs) for noncarcinogens have also been calculated where data permitted. These values are used for ranking reportable quantities; the methodology for their development is explained in U.S. EPA (1983a).

For compounds for which there is sufficient evidence of carcinogenicity, AIS and AIC values are not derived. For a discussion of risk assessment methodology for carcinogens refer to U.S. EPA (1980a). Since cancer is a process that is not characterized by a threshold, <u>any</u> exposure contributes an increment of risk. Consequently, derivation of AIS and AIC values would be inappropriate. For carcinogens, $q_1^*$s have been computed based on oral and inhalation data if available.

## ABSTRACT

In order to place the risk assessment evaluation in proper context, refer to the preface of this document. The preface outlines limitations applicable to all documents of this series as well as the appropriate interpretation and use of the quantitative estimates presented.

No data are available which address the potential carcinogenicity of this compound in humans. Limited *in vitro* mutagenicity evaluations have been negative. Only one cancer bioassay has been conducted. In this study 1,1,2-trichloroethane was carcinogenic in mice, but not rats by oral administration. Using the mouse data, a human $q_1^*$ of $5.73 \times 10^{-2}$ $(mg/kg/day)^{-1}$ was computed.

No data addressing the potential carcinogenicity of this compound by the inhalation route were located.

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Editorial review for the document series was provided by:

Judith Olsen and Erma Durden
Environmental Criteria and Assessment Office
Cincinnati, OH

Technical support services for the document series was provided by:

Bette Zwayer, Pat Daunt, Karen Mann and Jacky Bohanon
Environmental Criteria and Assessment Office
Cincinnati, OH

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## LIST OF ABBREVIATIONS

| | |
|---|---|
| ADI | Acceptable daily intake |
| AIC | Acceptable intake chronic |
| AIS | Acceptable intake subchronic |
| BCF | Bioconcentration factor |
| bw | Body weight |
| CAS | Chemical Abstract Service |
| CS | Composite score |
| ppm | Parts per million |
| STEL | Short-term exposure limit |
| TLV | Threshold limit value |
| TWA | Time-weighted average |

# 1. ENVIRONMENTAL CHEMISTRY AND FATE

The relevant physical and chemical properties and environmental fate of 1,1,2-trichloroethane (CAS No. 79-00-5) are as follows:

| | |
|---|---|
| Chemical class: | halogenated aliphatic hydrocarbon |
| Molecular weight: | 133.41 (Verschueren, 1983) |
| Vapor pressure: | 30.3 mm Hg at 25°C (Mackay et al., 1982) |
| Water solubility: | 4500 mg/ℓ at 20°C (Verschueren, 1983) |
| Log octanol/water partition coefficient: | 2.38 (Konemann, 1981) |
| BCF: | 21 (estimated from the equation of Veith et al., 1979) |
| Soil mobility: (predicted as retardation factor for a soil depth of 140 cm and organic carbon content of 0.087%) | <1.5 (Wilson et al., 1981) |
| Half-lives in | |
| Air: | 24 days (Singh et al., 1981) |
| Water: | 1.9 days (Zoeteman et al., 1980) |

The half-life of 1,1,2-trichloroethane in soil could not be located in the available literature; however, volatilization is expected to be the predominant loss mechanism from the soil surface. In subsurface soil, biodegradation of this compound is likely to be a slow process (Wilson et al., 1981). Based on the aqueous solubility and octanol/water partition coefficient, 1,1,2-trichloroethane is expected to leach into groundwater (Page, 1981; Wilson et al., 1981).

## 2. ABSORPTION FACTORS IN HUMANS AND EXPERIMENTAL ANIMALS

### 2.1. ORAL

Pertinent data regarding the absorption of orally administered 1,1,2-trichloroethane could not be located in the available literature. By analogy to other chlorinated ethanes, 1,1,2-trichloroethane is rapidly absorbed from the gastrointestinal tract (U.S. EPA, 1980b).

### 2.2. INHALATION

Pertinent data regarding the absorption of inhaled 1,1,2-trichloroethane could not be located in the available literature. By analogy to other chlorinated ethanes, 1,1,2-trichloroethane is rapidly absorbed following inhalation exposure.

## 3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS

### 3.1. SUBCHRONIC

3.1.1. Oral. Pertinent data regarding the subchronic oral toxicity of 1,1,2-trichloroethane could not be located in the available literature.

3.1.2. Inhalation. In an unpublished Dow Chemical Company study, Torkelson and Rowe (1981) exposed rats, guinea pigs and rabbits (numbers and strains unspecified) to 15 ppm (81.8 mg/m$^3$) 1,1,2-trichloroethane for 7 hours/day, 5 days/week for 6 months, or to 30 ppm (163.7 mg/m$^3$) 7 hours/day, 5 days/week for 16 exposures. No effects were noted on organ weight, hematology or clinical chemistry, but fatty changes were observed in female rats at the high dose.

### 3.2. CHRONIC

3.2.1. Oral. The NCI (1978) administered TWA doses of 46 or 92 mg/kg bw/day to Osborne-Mendel rats and 195 or 390 mg/kg bw/day to B6C3F1 mice (50 animals/species/sex/dose) by gavage, 5 days/week for 78 weeks, followed by a 13-35 week observation period. The corresponding controls consisted of 20 animals each. No dose-related, non-neoplastic changes were reported for either sex of either species.

3.2.2. Inhalation. Pertinent data regarding the chronic inhalation toxicity of 1,1,2-trichloroethane could not be located in the available literature.

### 3.3. TERATOGENICITY AND OTHER REPRODUCTIVE EFFECTS

3.3.1. Oral. Pertinent data regarding the teratogenicity or other reproductive effects of orally administered 1,1,2-trichloroethane could not be located in the available literature.

3.3.2. Inhalation. Pertinent data regarding the teratogenicity or other reproductive effects of inhaled 1,1,2-trichloroethane could not be located in the available literature.

## 3.4. TOXICANT INTERACTIONS

Traiger and Plaa (1974) reported that pretreatment with acetone or isopropyl alcohol (2.5 mg/kg bw by gavage) resulted in an increased hepatotoxic response to, and enhanced the effects of threshold doses of 1,1,2-trichloroethane in mice.

## 4. CARCINOGENICITY

### 4.1. HUMAN DATA

4.1.1. Oral. Pertinent data regarding the oral carcinogenicity of 1,1,2-trichloroethane in humans could not be located in the available literature.

4.1.2. Inhalation. Pertinent data regarding the carcinogenicity of inhaled 1,1,2-trichloroethane in humans could not be located in the available literature.

### 4.2. BIOASSAYS

4.2.1. Oral. The NCI (1978) treated groups of 50 male and 50 female B6C3F1 mice or Osborne-Mendel rats with 195 or 390 mg/kg bw/day (mice) or 46 or 92 mg/kg bw/day (rats), 5 days/week for 78 weeks by gavage. The mice were observed for an additional 13 weeks and the rats for an additional 35 weeks. There was no relationship between 1,1,2-trichloroethane treatment and the development of tumors in rats. All groups of treated mice had significantly ($p<0.01$) increased incidences of hepatocellular carcinomas. The incidences were 37/49, 18/49, 2/20 and 0/20 (males) and 40/45, 16/48, 0/20 and 2/20 (females) for the high-dose, low-dose, vehicle control and untreated control groups, respectively. The incidence of adrenal pheochromocytomas was increased in the high-dose groups (both sexes).

4.2.2. Inhalation. Pertinent data regarding the carcinogenicity of inhaled 1,1,2-trichloroethane in experimental animals could not be located in the available literature.

### 4.3. OTHER RELEVANT DATA

1,1,2-trichloroethane has been tested for mutagenicity in the Ames Salmonella typhimurium assay, both by the standard plate incorporation assay and by exposing the cells to vapors of the compound in a closed container (Barber et al., 1981; Simmon et al., 1977). Doses up to 158.9 μmol/plate,

a level toxic to the Salmonella strains used (TA1535, TA100, TA98), produced only negative results, both with and without the addition of rat liver S-9 preparation to provide metabolic activation.

### 4.4. WEIGHT OF EVIDENCE

Since 1,1,2-trichloroethane has only been demonstrated to induce liver tumors in one strain of mice in one experiment, the evidence for the carcinogenicity of 1,1,2-trichloroethane in animals is best considered "limited". Since no data are available regarding the carcinogenicity of 1,1,2-trichloroethane in humans, the chemical is best classified as a Group C compound - Possible Human Carcinogen, by applying the criteria for weight of evidence proposed by the Carcinogen Assessment Group of the U.S. EPA (Federal Register, 1984).

## 5. REGULATORY STANDARDS AND CRITERIA

The ACGIH (1980) has established a TLV of 10 ppm (~45 mg/m$^3$) and a STEL of 20 ppm (~90 mg/m$^3$), based on "the toxicological resemblance to symmetric tetrachloroethane and by analogy with the TLV for chloroform." The Occupational Safety and Health Administration has adopted this TLV as a general industry standard (Code of Federal Regulations, 1981).

The U.S. EPA (1980b) has estimated that a concentration of 6.0 µg/ℓ in ambient water will result in an excess lifetime cancer risk of $10^{-5}$.

# 6. RISK ASSESSMENT

## 6.1. ACCEPTABLE INTAKE SUBCHRONIC (AIS)

1,1,2-Trichloroethane is a chemical associated with liver tumors in mice and for which data are sufficient for calculation of a $q_1^*$. It is, therefore, inappropriate to calculate an oral or inhalation AIS for 1,1,2-trichloroethane.

## 6.2. ACCEPTABLE INTAKE CHRONIC (AIC)

1,1,2-Trichloroethane is a chemical associated with liver tumors in mice and for which data are sufficient for calculation of a $q_1^*$. It is, therefore, inappropriate to calculate an oral or inhalation AIC for 1,1,2-trichloroethane.

## 6.3. CARCINOGENIC POTENCY ($q_1^*$)

6.3.1. Oral. One study (NCI, 1978) has indicated that 1,1,2-trichloroethane is carcinogenic in B6C3F1 mice (see Section 4.2.1.). The U.S. EPA (1980b) has calculated a human $q_1^*$ of $5.73 \times 10^{-2}$ $(mg/kg\ bw/day)^{-1}$, based on the incidence of hepatocellular carcinoma in male mice (Table 6-1) and using a linearized multistage model.

6.3.2. Inhalation. Pertinent data regarding the carcinogenicity of 1,1,2-trichloroethane in humans or experimental animals exposed by inhalation could not be located in the available literature.

A complete data set for the derivation of the $q_1^*$ is presented in Appendix B.

TABLE 6-1

Incidence of Hepatocellular Carcinoma in Male B6C3F1 Mice Exposed to 1,1,2-Trichloroethane*

| Dose (mg/kg bw/day) | Incidence (No. Responding/No. Tested) |
|---|---|
| 0 | 2/20 |
| 195 | 18/49 |
| 390 | 37/49 |

*Source: U.S. EPA, 1980b

## 7. REFERENCES

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## APPENDIX A

### Summary Table for 1,1,2-Trichloroethane

| Carcinogenic Potency | Species | Experimental Dose/Exposure | Effect | $q_1^*$ | Reference |
|---|---|---|---|---|---|
| Inhalation | | | | ND | |
| Oral | mice | 0, 195 or 390 mg/kg bw/day | hepatocellular carcinoma | $5.73 \times 10^{-2}$ $(mg/kg/day)^{-1}$ | NCI, 1978; U.S. EPA, 1980 |

ND = Not derived

## APPENDIX B

### Cancer Data Sheet for Derivation of $q_1^*$

Compound: 1,1,2-trichloroethane

Reference: NCI, 1978

Species, Strain, Sex: mice, B6C3F1, male

Body weight: 0.33 kg (measured)

Length of exposure (le) = 546 days

Length of experiment (Le) = 637 days

Lifespan of animal (L) = 637 days

Tumor site and type: liver, hepatocellular carcinoma

Route, vehicle: oral, gavage

| Experimental Doses or Exposures (mg/kg/day) | Transformed Dose* (mg/kg/day) | Incidence No. Responding/No. Tested or Examined |
|---|---|---|
| 0 | 0 | 2/20 |
| 195 | 119.14 | 18/49 |
| 390 | 239.143 | 37/49 |

*Calculated to reflect treatment on 5 days/week and for 546 days of 637-day experimental period

Unadjusted $q_1^*$ from study = $4.4611191 \times 10^{-3}$ $(mg/kg/day)^{-1}$

Human $q_1^*$ = $5.73 \times 10^{-3}$ $(mg/kg/day)^{-1}$